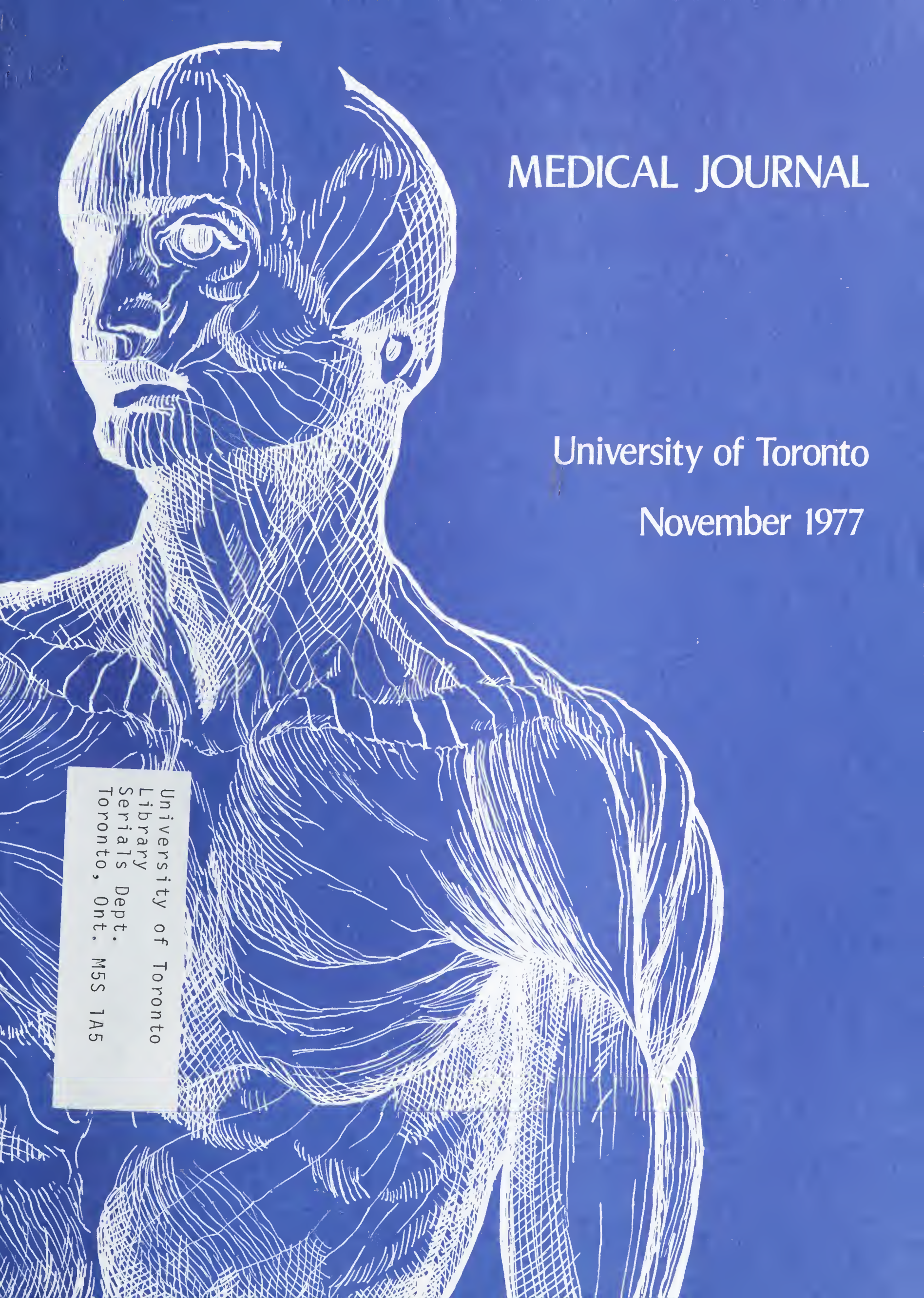




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CONTENTS

Editorial	3	
Medical Malpractice: An Overview	4	Peter Hayashida T. Edward Tichinoff
Further Studies on the Immunotherapy of Virus-Induced Osteosarcoma	6	Jay Wortsman
Blunt Dissection	13	
Alcohol and Drug-Related Deaths in Toronto and Vancouver: A Comparison	16	Jeffrey S. Leon
Book Review	22	
Feedback	22	

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- 1) All articles intended for publication should be submitted to the editors before the deadline which is usually six weeks before the release of the Journal.
- 2) Submitted articles should be typed (or legibly printed) double-spaced; single-sided on paper 8½" x 11" (letter size) and they should not be more than 15 to 20 pages (3000 to 4000 words).
- 3) Illustrations, graphs, diagrams and figures should be put on a separate sheet and identified by Arabic numerals. A list of explanatory legends for all the figures and diagrams should be put on another sheet. Do not write on the back of photographs.
- 4) References should be checked with the original sources, designated by Arabic numerals in the text, numbered in order of appearance and put on a separate sheet. Each periodical reference should include in order: the author's name and initials, title of the article, Journal name, volume number, page number and year. (eg., 1. Rosen HJ: Lumbar intervertebral disc surgery: review of 300 cases. *Can. Med Assoc J.*, 101: 317, 1969.)
- 5) The editors reserve the right to make such editorial changes as are deemed necessary to ensure correctness of grammar and spelling, clarification of statements and conformity to Journal style.
- 6) Acceptance for publication is subject to the understanding that such material is submitted solely to the Journal and will not be reprinted without the consent of both the editors and author.

From the Editor

Autumn crisp we are! With darting eyes and quickened paces we emerge from another long hot summer, chasing our not-so-new beginnings, all the while oblivious (or seemingly so) to the weight of another year of intensity-upon-intensity hanging over our heads. Forgive me for adding to it, but I can't remain silent about the disservice that was done you all at the Mad Hatter's Tea Party held in the Dean's Conference Room last September 26th (a.k.a., the Medical Assembly Budget Meeting). To mercifully condense a long and boring story, suffice it to say that the meeting resulted in a 13% decrease in monies allotted to the Journal this year (a 26% decrease from the Journal's 75/76 budget). Meanwhile, compared to 75/76 figures, the organizational allotments went as follows: MWUAA, up 87%; MAA, up 22%; Social Committee, up 27%; Orientation, up 33%; Auricle, up 29%; Camera Club, up 1000%; Open House, down 40%; Medical Society Special Projects, down 38%; Meds Lecture, down 17%; Class Funds, down 20%. Now, I don't care a tinker's damn how much of our student fee goes to sport and 'social activities', but when

it's doled out at the expense of the literary and research pursuits of the student body, something's rotten in Denmark. The Medical Assembly has lost its sense of balance and proportion; its priorities have become 'questionable', to be polite. I wonder how representative of its constituents the Assembly really is. Our elected reps have issued a **Catch 22** of sorts to the Journal in the Assembly's implication that if we were a 'better' Journal, we'd get more money allotted. Granted, to turn out a better Journal for less money is, to a degree, possible, and not

unreasonable. Last year's Journal was a solid first step on the road to that end. But let's be realistic! You can't have a better and better Journal for less and less money! In the end, as with all things, you get what you pay for. The Medical Assembly's official lack of interest and lack of support has us in an evertightening strangle hold. I decided to resign at least fifty times in the days following September 26th (once a day, on the average). Yet here I am, writing this protest when I should be reading Cardiology. (For the sake of argument, assume Cardiology to be the most fascinating subject in the world). Why? Perhaps because of the terrific encouragement I've received from fellow students, subscribers, and patrons. Perhaps because of the truly revitalizing effect of our 'fresh blood', our many keen Staffers from the class of 8T1. Perhaps because the Journal, as Canada's oldest, and, reputedly, best undergraduate medical Journal, is too good to let die before it's ever really matured. And finally, perhaps, in spite of all efforts to the contrary, and to my continual astonishment, I'm still an optimist, albeit, a despairing one at times. Welcome back!

Medical Malpractice: An Overview

Peter Hayashida, 8T1 and T. Edward Tichinoff

Medical malpractice suits are similar to other legal actions for damages brought about because of negligent conduct. Malpractice itself is defined as bodily harm incurred by virtue of neglect, abandonment or the omission or commission of certain actions which fall below the standard of care required of the average practitioner.¹ In principle the concept of malpractice is fundamentally just; a patient who believes that he has been injured by negligent or unskillful practice on the part of a physician seeks redress through the legal system. Yet in recent years the term has attained a high degree of notoriety. To many doctors, especially those south of the border, it has become an anathema; it has become like the sword of Damocles, a constant threat. To the public, malpractice has become part of contemporary vocabulary conjuring up images of inept doctors, scheming hypochondriacs and avaricious lawyers. The media have continually reported on the so-called American 'malpractice crisis' with its astronomical awards to claimants, soaring malpractice insurance costs, and strikes by physicians.

However, in Canada the same situation does not currently exist. We have no 'malpractice crisis'. Yet figures recently released by the Canadian Medical Protection Agency reveal an ominous increase in the number of malpractice claims in Canada. This leads one to ask such questions as: where is Canada headed in medical malpractice? will we follow the U.S. with its serious malpractice problem? what can we

do to avoid it? These are some of the questions we hope to answer in our brief overview of the medical malpractice issue.

The development of the malpractice crisis in the U.S. has been sudden and spectacular. Once uncommon in American tort law, more than 20,000 malpractice suits are now brought against doctors each year and the number is steadily rising.² One of the U.S.'s leading malpractice underwriters reports that the number of claims pending against doctors has risen from one in twenty-three in 1969 to one in ten today.³ Furthermore, in the past decade the average malpractice claim awarded has almost tripled, with many exceeding the million dollar mark. Consequently the malpractice insurance premiums paid by doctors have increased. For example, in Ohio the average premium has risen from \$2300 per annum in 1972 to approximately \$4000 today.⁴

These facts, however, are only the symptoms of the crisis. The real problem lies in the effects the malpractice activity has had on the practice of medicine in the U.S.. First of all, the cost to the consumer of medical services has risen in response to the higher insurance overhead of physicians. The malpractice phenomenon has also affected the quality of medical care; doctors have been forced to practice defensive medicine because of fear of possible malpractice suits which means that standard procedures are utilized rather than optional ones, and that unnecessary and wasteful tests are ordered, increasing the cost

...if one's expectations are not met, it must be negligence.

of health care. Another direct and detrimental effect is the fact that American physicians are generally far more reluctant to render emergency assistance in many circumstances. Two polls conducted in the U.S. showed that over 50%⁵ of the doctors questioned would not stop to assist an individual lying injured alongside the road, whereas a poll conducted in conjunction with the O.M.A. showed that over 90% of Ontario doctors would stop.⁶ The major reason cited by American doctors for not stopping was the fear of malpractice suits. This factor has resulted in several American state legislatures being forced to pass "Good Samaritan" statutes which remove the threat of legal action against those giving assistance in an emergency situation. Aside from these major effects, there is also a host of lesser problems: the added difficulties posed by high insurance costs for young doctors just establishing practices; the frustration and loss of time and money suffered by doctors in fighting malpractice suits, half of which end without an award or settlement.

The causes of the escalation in malpractice suits are more difficult to delineate than are the effects. Indeed, as the American Surgical Association has noted, "without any substantial change in the practice of medicine, there were twice as many malpractice suits in 1975 as in 1970, and the average jury award has increased sixfold."⁷ One factor in this development has been the increasing 'litigation consciousness' of the American people. Since the second World War the public has become more aware of its rights and has been exercising them more often in its courts.⁸ The change in the public's image of the doctor is also significant. The image is no longer one of the priest/healer but rather of a man of science, and this image of medicine is that of an "exact factual science" that **never** errs. Hence, *if one's expectations are not met, it must be negligence*. Also, with increasing specialization, doctors often provide impersonal care, with the result that the victim becomes more claim conscious as he reacts with animosity towards the indifference of mass medical practice.

However, a great deal of the fault may lie with the legal system as well. American lawyers work on a contingency fee basis: if they win a suit for a client they may receive up to 50% of the judgement, and if they lose, the client pays nothing. This not only encourages lawyers to enthusiastically take up suits that are not completely meritorious, but also brings out the gambling instinct in patients who are not fully pleased with their medical treatment. Moreover, the American constitution guarantees trial by jury in civil cases. This allows skillful, clever lawyers to play on juries' sympathies; they seem to continually favour the emotional appeal of the victim over that of the insurance company, regardless of the medical and technical merits of the case.

Thus has been the American experience in medical malpractice. In Canada we have relatively fewer malpractice suits and much less of a malpractice problem. This is reflected by the fact that in Canada most physicians (G.P.'s and specialists) pay \$200/year to the Canadian Medical Protection Association for malpractice in-

...the possibility of a malpractice crisis in Canada is considered unlikely by most experts.

surance, whereas in the U.S. specialists may pay up to \$30,000/year. The 76th. annual report by the C.M.P.A. indicates that legal action against doctors escalated rapidly from 1970 to 1974 and were unchanged in 1975 and 1976. Damages paid out by the C.M.P.A. in 1976 totalled \$2,664,103 compared with \$951,609 in 1975, illustrating the increase in malpractice claims.⁹ Although the number of cases has risen in Canada, it has not approached the magnitude of the American situation. These differences may be attributed to a number of factors. Firstly, contingency fees are not allowed in most Canadian provinces, including Ontario. In Canada a losing plaintiff may have to pay not only his own lawyers fees, but all court fees costs including most of the fees of the defendant. This is a disincentive to the instigation of irresponsible actions in our courts. Also in Canada, malpractice cases are usually tried before a judge alone, trial by jury usually being deferred on the grounds of complicated medical evidence.¹⁰ Another contributing factor is the climate of opinion in Canada, both among judges and the general public, which tends to be more sympathetic towards physicians than public and judicial opinion in the U.S..¹¹ Some researchers have also noted that 98.9% of health care costs for Canadians are borne by public compensation programs, which, along with the protective role of the C.M.P.A., have been significant factors in the lower incidence of malpractice suits in Canada.

Consequently, *the possibility of a malpractice crisis in Canada is considered unlikely by most experts*. Furthermore, there have been none of the adverse effects on Canadian health care as have occurred in the U.S.. That is **not** to say that Canada will never develop a significant malpractice problem. Given the public mood, changes in doctor-patient relationships and modes of patient care, claims against doctors and hospitals are almost certain to continue to rise. With the escalation of medical malpractice cases we may find that our system of handling malpractice may become as unruly and problematic as has the American system. However, **if** progressive measures are undertaken now, the entire problem **may** well be avoided.

Among informed commentators on the malpractice issue there has been a call for the removal of medical malpractice from the court system. As Justice Haines notes, "under the adversary system a medical malpractice case is one of the most difficult, expensive and unsatisfactory to all involved."¹² Doctors are generally reluctant to testify, they despise the courtroom procedure through which their professional competence is questioned and their wits and self-control badgered. The courtroom brings into conflict the methodology of the medical and legal professions. "The legal profession thrives on controversy, and the lawyer attempts to maintain his position, which he believes to be the truth, by argument and contention with opposing counsel. The physician, on the other hand, does not live by contention and controversy. His training and practice are in the clinical atmosphere of the laboratory and the hospital. He wants exploration of the case history and friendly discussion of all phases of the case. When all pertinent data are collected he correlates them and forms a judgement."¹³ Clearly the adversary system is not the best means of dealing with medical malpractice. **Is there a better way of resolving these matters?**

(continued on page 14)

Further Studies On the Immunotherapy Of Virus-Induced Osteosarcoma

Jay Wortsman, 7T9

Human osteosarcoma is a malignant neoplasm derived from cells of the osteoblastic series and therefore has the potential to form bone.¹ It is a not uncommon disease that is, after plasma cell myeloma, the most frequently occurring primary malignancy of bone.² It is encountered most often between the ages of 10 to 20 years,³ the years of adolescent bone growth, showing a marked predilection to develop at the metepiphyseal zones, the very site of greatest osteoblast activity.⁴ The five year survival prior to 1972 was 20%, with the majority of patients dead within 21 months of the onset of symptoms,^{2,3} within six months when pulmonary metastases are demonstrable.^{3,5} The cause of death is "malignant cachexiaemaciation and asthena with or without amenmia".⁵ The most common autopsy finding is multiple, bilateral lung metastases(90%), with pleural effusions and some degree of lung collapse(50%).⁵

The treatment of osteosarcoma is approached by surgery, radiotherapy, chemotherapy and immunotherapy. In the United States and most other countries, the treatment of choice is amputation of the primary site.⁶ Its success rate is the historical 20% five year survival rate.⁶ In Britain, the additional treatment of radiotherapy to amputation raises this figure insignificantly to a 24% five year survival.⁴ It is felt that radiotherapy is of little aid in the treatment of osteosarcoma because (1) the very

high resistance of the tumour to radiation^{3,7} (2) osteosarcoma is a highly metastatic disease which at the time of diagnosis may already have metastasized to lungs, liver and other organs. To quote Stein, "since osteosarcoma is a systemic disease and because of the brief period of symptoms and rapid onset of metastases following therapy, any further improvement in survival statistics will have to be the result of additional therapy directed to the systemic spread of disease."³

Significant chemotherapy directed against osteosarcoma can be said to have begun in 1972.⁸ Such agents as methotrexate⁹ and adriamycin¹⁰ were given separately to small groups of patients. More recently, multidrug chemotherapy treatments have been employed with considerable success, one study¹¹ reporting of a metastatically free halt in the progression of osteosarcoma in two-thirds of the patients participating in a five drug chemotherapy program. However despite these encouraging results findings, Jaffe¹² cautions against interpreting these results with only one or two years duration and adds that, under chemotherapy, the time interval between diagnosis and appearance of lung disease may be lengthened for late metastasizers.¹³ We will note here that chemotherapy suffers from several adverse features not the least of them are (1) cytotoxicity by the anti-tumour drug to normal regenerating tissues(specifically the gastro-intestinal and lymph-haemopoietic

systems) with consequential increased susceptibility to secondary diseases. (2) the inherent ability for the anti-tumour drug to induce a new neoplastic growth in either non-cancerous or cancerous tissue.¹⁴ (3) uncertainty of the drug's ability to eradicate tumour or simply to retard its neoplastic growth.⁸ (4) potential for tumour cells to mutate(naturally or drug-induced) and become resistant to cytotoxic chemotherapy.

This leads us then to the clinical approach by immunotherapy. In 1963, clinical studies were conducted where normal lymphoid tissue, as single cell suspensions, was transplanted into patients with advanced cancers.¹⁵ The rationale was that this added lymphoid population would aid in the immunological combat of the cancer. It has previously been shown that allogeneic¹⁶ and xenogenic¹⁷ spleen cells from normal animals could temporarily halt the growth of experimental tumours when these cells were injected into the test animals.

The donor cells set up a graft-versus-host reaction(GVHR) and a reciprocal host-versus-graft reaction(HVGR) is mounted by the host against these donor cells. The HVGR is liable to destroy the transplanted cells unless the host's immune system(cell-mediated) is rendered inactivated, something one wouldn't do in the cancer situation. Alternatively, one could inject parental cells, which in the case of pure-bred strains of test animals, would not evoke a HVGR in the F1 hybrid, as it would be incapable of

detecting either of the parental cell lines as foreign. This classical GVHR experiment then would allow one to dissect the attributes of a GVHR from a concurrent HVGR.

It is now evident, due to the severity of the disease and the low survival rate, that an animal model of osteosarcoma would be of immense benefit. Such a model has been induced by radionuclides, chemical carcinogens and viruses. Radionuclides have the inherent disadvantages to the experimenter of a long latency period and an artificial, laboratory induced form of oncogenesis since less than 5% of osteosarcomas are thought to originate by radionuclide means.¹⁸ Chemical carcinogens have the poor laboratory feature of exhibiting varied tumour-specific-antigens(T.S.A.) on the plasma membrane making results difficult to interpret and duplicate. Viruses do not have this drawback, as it is felt that virally induced neoplasms have similar if not identical T.S.A.'s within any given strain of animal and possibly across strains. It is also presently felt that human osteosarcoma is associated with a virus.^{18,19,20,21}

Using then the Maloney Murine Sarcoma Virus, an animal model of osteosarcoma was investigated which "resembled the human tumour

closely in terms of site of formation, histogenesis, growth pattern, osteoid production, destructiveness and ability to metastasize to the lungs".²²

It has the advantages of having a short latency period (10 days), is easily induced in neonatal rats with a high degree of take(approximately 90%) and it can be serially passaged as single cell transplants within the same strain.²³

The following experiment was work undertaken by the author, during the previous summer, in the orthopaedic research laboratory at the Mount Sinai Hospital in Toronto. This experiment formed the logical progression of work already conducted in this laboratory. In this study we wish to determine what effect the effect of amputation of the primary tumour and graft-versus-host reaction was on the virally induced model of osteosarcoma.

Materials and Methods

The animals used in this experiment were the pure-bred rat strains AC1, Lewis/Mai and Brown Norway. The test animal was the four to fourteen week old F1 hybrid of male AC1 x female Lewis/Mai strain.

Primary and Secondary Tumour Induction

Maloney Murine Sarcoma Virus of a titre of 5.7 log F.F.U./ml was stored at -70° C until time of use. A standard quantity of .025 ml of this virus solution was injected into the tibial bone marrow cavity of one day old F1 hybrid rats. When the tumour had grown sufficiently large at the primary site of induction, the animals were sacrificed and the tumours excised under semi-sterile conditions. The tumours were cleaned of necrotic material in a normal saline bath and transplanted as small(2x2x2 mm³) pieces into the subcutaneous compartment on the dorsum of several adult F1 hybrid rats. When tumour growth in these rats was of sufficient size, the animals were sacrificed and the tumour excised semi-serilely, and cleansed of necrotic material in a normal saline bath. The tumour was then finely minced in alpha media* and then subjected to enzymatic digestion with trypsin and collagenase. The single cell suspension attained after washing, centrifugation and re-suspension is called the master stock(1st. passage). Master stock is frozen at -70° C in a 10% solution

* A minimal essential nutrient media.

EXPERIMENT A

STRAIN	CONTROL						TEST					
	DAY	#/Gr.	$\bar{x}$	s.d.	s.e.	s/d.o.d	DAY	#/Gr.	$\bar{x}$	s.d.	s.e.	s/d.o.d.
ACI	-3	2	43.5	12.02	8.50	3+	-3	3	30	3.61	2.08	(only 3)
	0	2	28.3	9.19	6.50	(only 2)	0	3	27	2.65	1.53	0+
	+3	3	37.3	10.26	5.93		+3	3	32.7	2.09	1.90	0+
	+7	3	27.3	2.31	1.33		+7	4	39	5.03	2.52	
Brown Norway	-3	3	38.7	9.02	5.21		-3	3	28	1.73	1.00	
	0	2	23.5	3.54	2.50	1s	0	2	30.5	.71	.71	1s 52+
	+3	3	30.7	2.52	1.45		+3	4	25.75	3.30	1.65	
	+7	1	59.0	-	-	2s	+7	4	28.75	7.41	3.71	
LEWIS/MAI	-3	3	25.3	.59	.34		-3	4	30.75	4.50	2.25	
	0	3	27.7	5.03	2.91		0	4	29.25	7.63	3.82	
	+3	3	28.3	2.52	1.45		+3	4	28.75	5.32	2.66	
	+7	2	21	-	-	1s	+7	3	24.3	3.51	2.03	1s
F1	-3	2	32.5	2.12	1.5	1s	#/Gr.= number per group for average $\bar{x}$ = average s.d.=standard deviation s.e.=standard error s/d.o.d.= survivals, or day of death					
	0	3	28.7	4.51	2.60							
	+3	2	32.5	13.44	9.50	1s						
	+7	2	25	1.41	1.00	52+						

of dimethylsulfoxide in alpha media at cellular concentration of 1×10^6 cells/ml.

To grow the second passage for the whole of the experiment, several vials of master stock tumour cells were thawed at room temperature, washed in alpha media, centrifuged and reconstituted in normal saline and injected into the subcutaneous compartment on the dorsum of several rats adult rats. Tumour was harvested as above and frozen as until needed. This is the second passage or working stock.

Harvesting Lymphoid Tissue and Induction of GVHR

Part A:

Parental(Lewis/Mai and AC1), third party(Brown Norway) and syngeneic(F1 hybrid) lymphoid cells were all harvested identically. Donor animals were anaesthetized with ether, and their spleens excised under semi-sterile conditions. The spleens were cut into pieces upon excising and placed into alpha media on ice for transport. The spleens were then mashed through wire screening to attain a single cell preparation. The cells were centrifuged, re-suspended in .84%

NH_4Cl to lyse the red blood cells and re-washed three times in normal saline. For innoculation into the peritoneal cavity, (I.P.) to induce the GVHR, a dosage of 50×10^6 viable lymphoid cells in .5 ml saline was used.

Part B:

Part B is identical to part A except that all donor animals had been pre-sensitized to F1 hybrid tumour 12 days prior to spleen harvesting. This was done by injecting 1×10^6 viable F1 hybrid tumour cells into the tibial marrow cavity of donor animals.

Tumour Inoculation

Tumour inoculation consisted of preparing second passage tumour, already stored at -70°C , as discussed earlier. Tumour cell suspensions were set at 20×10^6 viable F1 hybrid tumour cells/ml. The standard inoculum was .05 ml. These were all done into the tibial bone cavity.

Amputation

Within an hour of the tumour transplant into the tibial bone marrow cavity, the rat was anesthetized with ether and the leg which received the tumour inoculum amputated. This was achieved by locating and ligating the femoral vein and artery as they passed in and out of the femoral canal. The femur was then fractured at about the proximal third from the diaphysis. Bone fragments were removed, muscle and sheath sewn over the stump and skin opposed and stapled together over the stump with nine mm. autoclips. The rat was observed until recovery from anaesthesia.

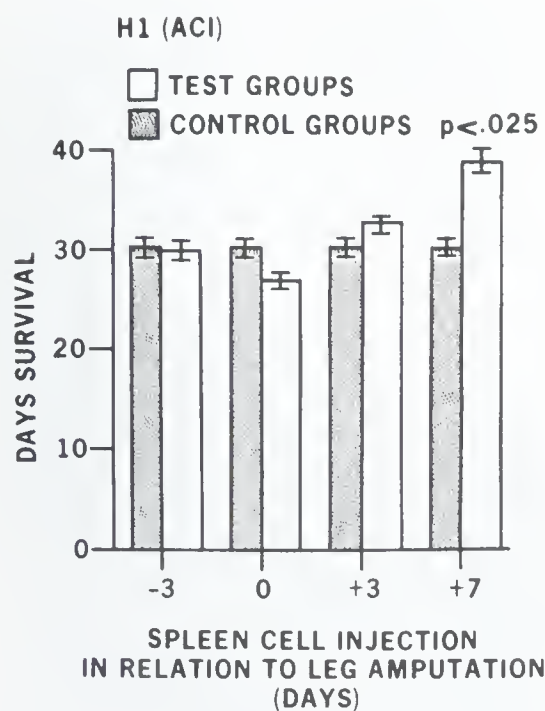
Experimental Set-Up

Recipient(test) animals of spleen cells and F1 hybrid tumour intratibially(I.T.) were all four to fourteen week old F1 hybrid rats. Donor animals for spleen cells were AC1, Lewis/Mai, Brown Norway and F1 hybrid.

Day zero is said to be the day of F1 hybrid tumour transplantation(I.T.) and amputation. Spleen cell injections were given on day -3, day 0, day +3 or on day +7 such that any

EXPERIMENT B

STRAIN	CONTROL						TEST					
	DAY	#/Gr.	$\bar{x}$	s.d.	s.e.	s/d.o.d.	DAY	#/Gr.	$\bar{x}$	s.d.	s.e.	s/d.o.d.
ACI	-3	3	-	-	-	3s	-3	4	31	4.08	2.04	
	0	3	36	7.55	4.36		0	4	25.75	6.55	3.28	
	+3	3	31	-	-	2s	+3	2	33.5	3.54	2.50	1s 12+
	+7	3	30	-	-	2s	+7	4	29.75	6.08	3.04	
Brown Norway	-3	3	36	1.41	1.00	1s	-3	3	27.67	2.31	1.33	(only3)
	0	3	28	2.83	2.00	1s	0	4	25.25	4.03	2.02	
	+3	3	-	-	-	3s	+3	4	30.5	5.26	2.63	
	+7	2	37	-	-	2s	+7	4	26.25	1.89	.95	
Lewis/Mai	-3	3	34	-	-	2s	-3	4	34	4.76	2.38	
	0	3	32.5	.71	.50	1s	0	4	24.25	3.77	1.89	
	+3	3	29	-	-	2s	+3	4	28	3.16	1.58	
	+7	3	46	-	-	2s	+7	4	33	4.90	2.45	
F1	-3	2	32	1.41	1.00	(only2)	#/Gr.=number per group for average $\bar{x}$ = average s.d.=standard deviation s.e.=standard error s/d.o.d.= survivals, or day of death					
	0	3	30.3	2.52	1.45							
	+3	3	-	-	3s							
	+7	2	31	1.41	1.00	1s						



group of animals received only one inoculum(i.e. AC1 rats day -3 meant F1 hybrid animals were given 50×10^6 viable AC1 spleen cells I.P. and then received the F1 hybrid tumour I.T. and amputation four days later, i.e. on day 0).

There are four rats per group in the test animals for a total of ($4 \times 4 \times 3$ or) 48 F1 hybrid rat recipients. (see charts "test").

For control, the same procedures were held but there were three alterations. (1) instead of four animals per group there were only three. (2) an extra set of animals was added, namely F1 spleen cell donors, also for days -3, 0, +3, and +7. (3) AC1, Lewis/Mai and Brown Norway spleen cells were lethally irradiated with 950 R just prior to injection into the recipient control animals I.P. The total number of rats used was ($4 \times 3 \times 4$ or) 48 rats.

Results

Data for the experiments, parts A and B, has been processed and tabulated on charts A and B respectively. For purposes of processing results, rats dying within 14 days after amputation are considered to have died from reasons other than from tumour(i.e. surgical trauma, haemorrhage, bacterial infection, etc.). Those rats surviving greater than 120 days are labelled on the charts as "S", signifying long term survival.

Part A

Results for experiment A are plotted on histograms H1, H2, and H3 for donor species AC1, Brown Norway and Lewis/Mai respectively.

The control group for all three histograms (30.3 ± 1.49 S.E. in days) is found by combining the results of the control populations for the strains AC1, Brown Norway and Lewis/Mai(29 rats) over all the days. A Student's T test was performed against the control for all three donor strains. Only day +7 in the AC1 group yielded significant results.

Part B

Results for experiment B are plotted on histograms H4, H5 and H6 for donor species AC1, Brown Norway and Lewis/Mai respectively.

The control group for these three histograms was arrived at by combining the results of the control population for part B(for those rats that died) of all non-isogeneic(to F1 hybrid) strains over all the days(33.3 ± 2.14 S.E. in days) analogously to part A. No significant results were found when the test animals were compared to the control value.

Discussion

We will first discuss our method of arriving at control values in parts A and B.

In part A, a Student's T test was performed on the F1 hybrid donor group and the derived control mean(of non-isogeneic donor animals) for each separate day(i.e. -3, 0, +3, +7). No significant difference was found. We maintained the distinction of F1 hybrid cells from the rest of the control groups simply because the F1 cells were viable and the other donor cells lethally irradiated prior to injection. We then gathered the days over all the pure-bred strains to arrive at the control mean. This value was compared against the average value of each day of each control strain, F1 hybrid included, to insure that this

* with the exception of day +7 Brown Norway. This is discarded since it survived unduly long while its group mates were long-term survivors. We consider this an experimental error.

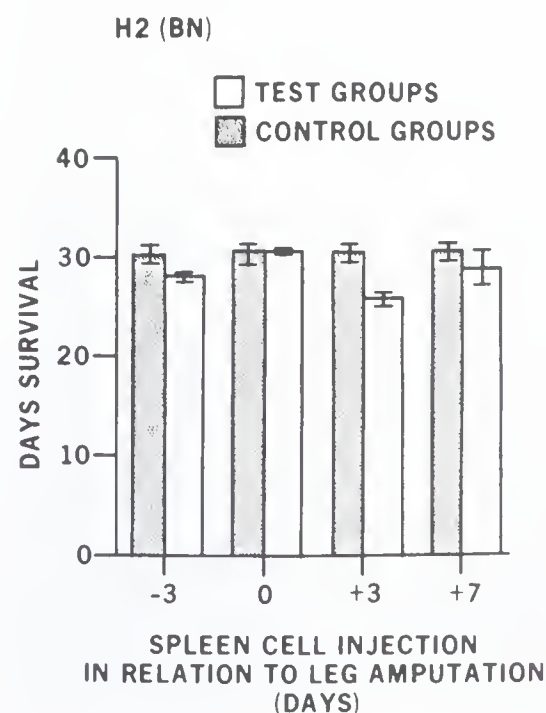
control mean was not significantly different from original control values from each strain. No significant difference was shown by the Student's T test. We, therefore, felt confident in justifying this pooling of results over all strains, since it was believed that no effect should come from the lethally irradiated spleen cells anyway, regardless of strain origin.

Part B's control value went through analogous processing and testing. The main difference here was that the common mean collected from all the days(i.e. -3, 0, +3, +7) showed significant differences when compared against the F1 hybrid control cells($p < .025$ on day -3, $p < .10$ day +3, $p < .10$ day +7). We will come back to this finding further on in the discussion.

Experiment A

Previously Czitrom²⁴ et al was able to show GVHR altering survival values in a positive manner on days -3, 0, +7 for F1 hybrid rats treated with 50×10^6 viable AC1 spleen cells(the only parent tested) and on day 0 for F1 hybrid animals treated with Brown Norway cells at the same dosage. His contention was that AC1 spleen cells induced a graft-versus-host reaction that helped retard tumour growth and so prolonged rat survival time. He also found third part(ie. Brown Norway) cells to enhance survival times, but only on day 0, a fact he attributed to the direct anti-tumour action of these immunocompetent cells.

In this experiment AC1 donor cells

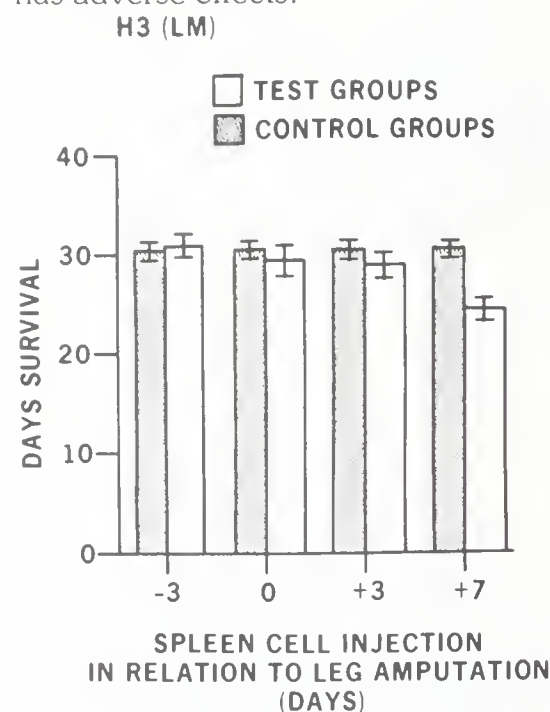


show no significance difference on days -3 and +3, an adverse effect on day 0 and the only significant statistic, a marked enhancement on day +7. We'd like to put forward the explanation that the trauma of surgery masks the possible benefits derived from GVHR so that it would require a period of time before GVHR therapy could be started on the animal after surgery. To support this stand, we point to the "trend" in survival starting from day zero. In fact Czitrom's work shows that animal survival is more significant on days 0 and +7 ($p < .005$) than on day -3 ($p < .01$) so that we could argue that surgery alone has simply "lowered" survival values uniformly and the best time to initiate a GVHR is, as in Czitrom's paper, after day +3.

In consideration of the other parental strain (Lewis/Mai) we observe a reversal of survivability, that is, progressing from day -3 to day +7, the shortening of tumour induction time and decrease of the animals survival time. This is a curious and contradictory result. An experiment was therefore devised whereby 50×10^6 viable Lewis/Mai spleen cells were injected I.P. into six normal four month old F1 hybrid rats. At 12 days these animals were sacrificed as well as six litter mate controls. Spleen weight to body weight ratios (spleen index) were taken as a measure of severity of GVHR, for both test and control animals. By comparing the spleen index of the test animals to that of the control animal, splenomegaly could be quantitatively assessed. Anything above 1.30 is considered significant. AC1 donor strain cells induced a previous spleen index comparison of $1.64 \pm .22$ S.E.²³

We took the spleen index of the control animals and dropped the highest and lowest values. A linear regression was then plotted of spleen weight to body weight for these control values. A correlation coefficient of .68 was achieved. Using this line for an estimation of normal spleen weight for a given body weight, we took spleen test indices and compared them against estimated control spleen indices. We obtained results with a minimum of 1.36 to a maximum of 3.03 times normal ($\text{mean } 2.46 \pm .26$ S.E.). This

would indicate a very strong GVHR. We would like to postulate the seemingly counter argument that due to the severity of the GVHR, the animal actually succumbs faster and at the point in time where a milder dose of spleen cells would be beneficial. This excessive reaction at the stated dose, however, actually has adverse effects.



It is well recognized that the severity of the GVHR depends at least upon (a) the antigenic constitution of host and donor (b) the number of immunocompetent cells injected.¹⁶ If either the Lewis/Mai lymphoid cell line presents a stronger anti-genic reaction against the AC1 markers of the F1 hybrid cells (than the corresponding AC1 lymphoid reaction against the Lewis/Mai markers on the F1 hybrid cells) or if the absolute number of Lewis/Mai lymphoid cells transplanted survive longer in vivo in the F1 hybrid than the AC1 cells, then we would have a more severe GVHR induced by the Lewis/Mai, than AC1 cells. It can be easily seen then that a greater GVHR, as indicated by the increase spleen index, could compromise the F1 hybrid's ability to resist the tumour growth when it is already suffering from the GVHR induced secondary disease.

Little discussion can be derived from Brown Norway donor cells. The survival is not increased and in fact is decreased to some extent. This is not a surprising result as the F1 hybrid immune system is now called upon to fight the tumour cells as well as allogeneic cells. A minor point we shall draw attention to is

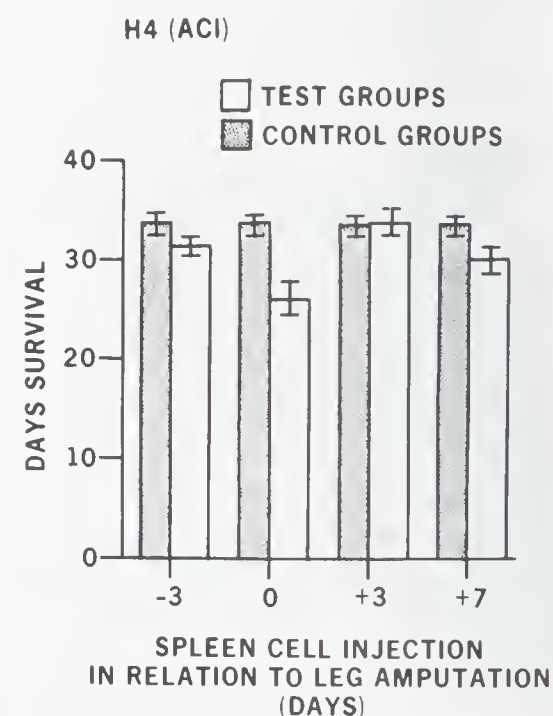
that only on day 0 is no effect seen.* We will argue that on this privileged day the allogeneic cells might be aiding the F1 hybrid immune system in combating the tumour or at least not provoking too severe an immune response as to "draw attention" away from tumour attack. This is analogous to Czitrom's finding with the explanation, again, that the surgery is masking any possible benefit derived from this allogeneic transplant and so lowering survival values further than those which would occur without surgery.

Experiment B

Originally, on devising the experiment, part B was to be used as a control, to see if GVHR alone, with amputations was effective in delaying or even obliterating tumour growth. In this experiment the donor spleen cells were pre-sensitized to the F1 tumour line 12 days prior to harvesting. Comparison, then should be of experiment B test animals to experiment A test animals.

Comparison, day for like day, strain for like strain, shows no consistent findings. There are some significant results by Student's T test but only one extreme. This is the Lewis/Mai day +7. In experiment B, the animals survive an average of 33 days which is a significant enhancement. The T test result for the +7 day Lewis/Mai of experiment A and experiment B

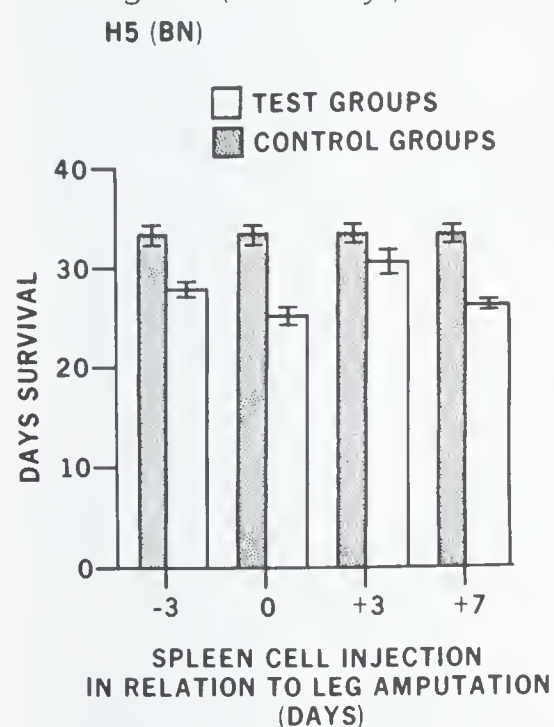
* Results are derived from two rats' deaths. A third rat lived 52 days before dying and a fourth rat became a long term survivor. These later two results we believe to be experimental errors.



comparision yields a $p < .025$. No explanation for this result will be forwarded other than the belief of a spurious result.

A similar but oppositely directed result is seen in the AC1 day +7 group where the two experiment's comparison yields a $p < .05$.

Controls were of course provided whereby irradiated(950R) spleen cells from the three strains were used after having first been pre-sensitized to F1 tumour 12 days prior to harvesting. Control animals developed into two categories: Those that succumbed to the tumour and long term(>120 days) survivors.



Comparing the test animals against the animals that died we find in general the control group survived longer than the test an group, in some cases markedly so(see H5). This is an unexpected and curious finding. We shall dissect the discussion into the three strains of rats.

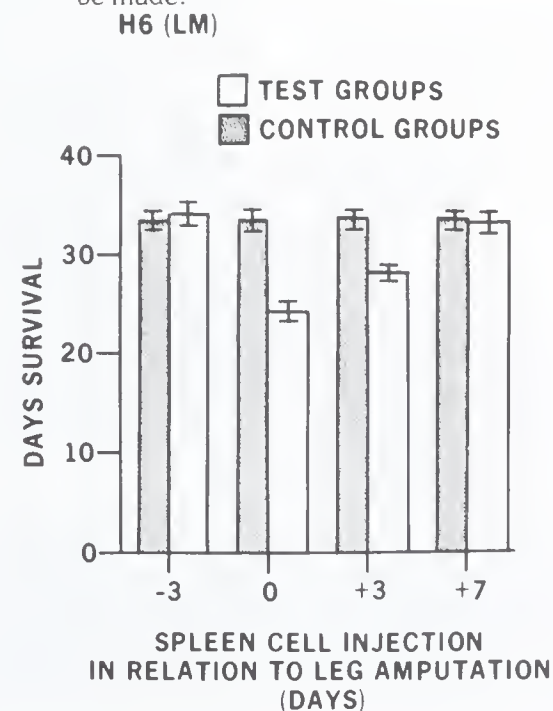
Histogram H4 shows that the AC1 donor treated animals dying faster or at the same time as the control animals. Evidently GVHR, if it is expressed here has not had any positive effect until we compare against histogram H5, the allogeneic cell. Here in every case, tumour growth and animal death is greatly accelerated as compared with control animals. It would seem then that the mild GVHR supposedly expressed in H4 has slowed tumour growth. We can not compare with H6, the other parental strain, except to say that in this case too(as in H3) the GVHR did not retard tumour growth and may in fact have aided it.

In light of the fact that the control F1 cells were injected as viable spleen cells whereas the other control cells are lethally irradiated and in light of the previous discussion regarding proper controls for experiment B we feel that the comparison between F1 and other species, though noted earlier in results, is not really valid and so no further study of them is demanded.*

Before discussing the matter of long term survivals we should like first to offer explanation why the experiment did not prove to be as fruitful as originally planned.

In preliminary work leading up to this experiment, Czitrom tried amputation on days 0, +1, +3 after tumour transplantation. The survival of the animal did not seem to matter. Perhaps this is because by the method of tumour transplantation, injection of tumour into the marrow cavity, causes enough systemic dispersion in the blood as to make any attempt at decreasing the tumour load by amputation of the primary, futile. An attempt to alleviate this situation was made by cutting back the initial tumour inoculum of 10^6 cells to 10^5 . The result was that the animals did not always get tumour, and those that did took longer to die from the tumour. Clearly the constraints of the tumour growth dictated the higher tumour load and amputation, for whatever purpose it can serve, as soon after tumour transplantation as possible.

* We note that F1 hybrid control animals day +3 all survived. This may be a significant finding or an experimental error. Further study must be done before comment can be made.



We now come to the most interesting, and frankly, unexpected result of the experiment, the matter of animal survival.

A quick scan of the tabulated chart will reveal random survival of animals in part A and in the test of part B. These we attribute to experimental error. Perhaps insufficient number of tumour cells were injected in some cases. Perhaps amputation cleared the animal of more of the tumour load than in the average case, perhaps more or less spleen cells than calculated were injected I.P. to aid the animal directly or by GVHR. Any or all of these things could account for animal survival in such small groups as one or two animals per group(remembering that the size of a group was only three(as in a control) or four(as in test) animals).

But when we look at part B control we find about 60% survival in the parental and allogeneic donor strain animals. We suggest a transfer-factor like phenomom is occurring.

Transfer factor, first described in 1955 by Lawrence²⁵ is a dialyzable extract of sensitized lymphocytes that is unaffected by ribonuclease or deoxyribonuclease. Transfer factor is capable of transferring the immunological memory of the donor(i.e. delayed hypersensitivity), is non-antigenic itself and can be stored for long periods of time. It is thought to work by replicating in the recipient, causing conversion of non-sensitized lymphocytes into sensitized, reactive ones.²⁶

In an analogous experiment, conducted in 1969, Dupuy et al was able to passively transfer delayed hypersensitivity to purified protein derivative in two-thirds of normal guinea pigs by three daily injections of a small volume of plasma from B.C.G.-immunized, x-irradiated(800-1000 rads) donors.²⁷

Neither plasma from non-irradiated but B.C.G.-sensitized, nor plasma from irradiated but non-sensitized donors could transfer this sensitivity to normal recipients. They could also transfer this sensitivity by incubating normal spleen cells with plasma taken from irradiated, pre-sensitized donors. They could not remove this property by repeated washings. They finally concluded that thymus-dependent factor(s)

responsible for cell-mediated immunity can be released from cells by x-ray and absorbed by normal lymphocytes by incubation *in vitro*. We believe that our experiment might contain the *in vivo* analogue of sensitizing normal spleen cells with x-irradiated pre-sensitized lymphocytes.

To support our view of transfer-factor involvement we draw upon human experiments carried out on patients with extensive and advanced osteosarcoma. In one study²⁸, 12 unrelated patients with osteosarcoma had some of their tumour taken at the time of amputation and implanted into a recipient patient also with osteosarcoma. Fourteen days later, recipients underwent leukophoresis. The number of mononucleocytes then transferred to the original donors was of the order of 10^9 cells.

The result of this report was that a delayed hypersensitivity could be demonstrated against osteogenic sarcoma. Their preliminary work show three patients responding well to treatment though they do not claim that treatment is solely responsible.

In a more recent paper by Levin²¹ et al, transfer factor itself derived from normal household contacts demonstrating cytotoxic (cell-mediated) reactivity to osteogenic sarcoma, was used. Transfer factor was prepared by freezing and lyophilizing white cells obtained by leukophoresis. This lyophilized material was then reconstituted in distilled water, dialyzed, lyophilized again and reconstituted in water, in doses of about 10^9 leukocytes.

Results were that an increase in cell-mediated cytotoxicity to the tumor occurred as suggested by increased lymphocytic infiltrate into the tumour and increased pain and edema (signs of lymphocytic involvement). In the five of eighteen patients who were clinically tumour-free at the beginning of this immunotherapy problem, all remain so (12-24 months after diagnosis). The rest remain stable or are deteriorating. They suggest that this form of therapy is suitable for clinically metastatic-free patients.

In our experiment, we hazard then to suggest that a transfer-like phenomenon may account for the

increased survival in the control rats treated with pre-sensitized, x-irradiated lymphocytes. If this is true, it would suggest that transfer factor derived from allogeneic cells is comparable to that derived from semi-syngeneic (parental cells).

We caution the reader that these results are preliminary and due to the small number per group, need repetition of results.

Summary

An experiment was devised to express the efficacy of adding surgery to the therapy of graft-versus-host disease previously shown to be beneficial.²¹ Results show at the dosages of tumour and spleen cells injected, a significant improvement could only be shown on day +7 in AC1 donor strains cells.

Part of the experimental set-up inadvertently provided released of a transfer-factor like phenomenon which may have rescued some 60% of rats with osteogenic sarcoma. This serendipitous finding may provide an animal model for the study of transfer factor on tumour growth. □

I wish finally to express my gratitude and sincere appreciation to Dr. A. Czitrom, Dr. F. Langer and Dr. A. Gross for the opportunity to participate, for their guidance throughout, and for their encouragement to conclude.

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Should patients be given access to their medical records?

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Giving patients access to their own medical records could serve several important functions. It would enable the patient to take an active part in his/her health care; encourage ongoing doctor and patient education, and be a first step in improving the doctor-patient relationship. Several ethical issues could also be simplified if the patient was to assume a more active role, in terms of the receipt of information. Perhaps most important, is the increase in personal responsibility for one's own health care that would be initiated and symbolized by the reading of one's chart.

Understood in the context of patient access to medical records is that the records would be interpreted with the help of members of the health care team (MDs, nurses, OTs, physio, social work, etc.). In the course of these discussions on prevention, diagnosis and management of disease, the patient's role in prevention and treatment would be increased. The patient would be aware of what is to be done, why, the alternatives, as well as his/her role in the process.

Obviously there are people to whom such responsibility would be irrelevant. Patients with psychiatric disorders or an inability to perceive and interact with their environment might be reasonably excluded from this access.

The doctor-patient relationship can be among the most intense factors in a patient's perception of his health and disease. Yet the Ontario Health Disciplines Board has found many doctors to be arrogant and unresponsive to the needs of their patients. "This health care system treats patients like little blocks of wood," said a well-educated and articulate patient on being informed that he was being transferred to yet another service in the hospital.

"Doctor" means teacher. It would be a major advance in the prevention and treatment of disease, if the medical profession could take the time to explain fully the significance of lifestyle, environment, and what is known of the natural history of disease and their personal relevance, to interested patients. In this way the doctor could maintain the role of resource person and personal advisor, and relinquish the role of super-human decision maker.

At present patients may modify lifestyle "for the doctor's sake"--not as means to better health for themselves. Increasing personal responsibility for these changes could make a positive contribution toward the situation, as described in Lalonde's *A New Perspective on the Health of Canadians*: "There is little doubt that

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The price paid for giving patients access to their medical records would be too high. There is no question that doctor-patient relations need to be improved, or that patients need to feel more responsible for their own health. But these are not at all inevitable consequences of giving patients access to their records--and some of the consequences that are inevitable, are very undesirable.

Communication, education and personal interaction would need to be improved if the chart were ever to be anything except a maze of numbers and medical jargon to the patient. Unfortunately, access to records can be legislated and enforced, while improved doctor-patient relations cannot. Many ethical physicians would be motivated to give all the background and counselling necessary to an understanding of the chart, even under present constraints of time and money.

Many others would not. Even those with the best of intentions might inevitably slide into a pattern of giving a quick talk on the differential diagnosis and prognosis and handing over the chart, with an encouraging suggestion that the patient "call if there's anything you don't understand." The perfectly-understood chart would encourage a sense of responsibility and trust; the imperfectly-understood chart would almost certainly result in misinterpretation and needless fear.

The function of charts would change significantly the moment the patient entered the chain of readers. Content would change to reflect the new function--and though the change might be in the direction of simplicity and full explanation, if one goes on the example of bedside communication between physicians, it is more likely to result in a positive flourishing of new jargon, of neo-Latin and euphemisms like "increased mitotic figures" whose interpretation would be left up to the patient's personal physician. A dictionary could be a very cruel counsellor if the physician took this task lightly.

A second possibility is the development of a dual system of record-keeping: one set for the physician, and another possibly different set for the patient. Consult notes, too, might change. An oral report over the phone might become the true communication between physicians, while an incomplete letter entered the file for interpretation to the patient at the personal physician's discretion.

These arguments all seem to assume the physician has something he wants to conceal from the patient. A major thing private records have concealed from patients, is the complexity of interpretation necessary in evaluating even apparently simple disorders. Simply by

future improvements in the level of health of Canadians lie mainly in improving the environment, moderating self-imposed risks and adding to our knowledge of human biology."

In addition, changing a health care system that now allows for little two-way communication between patient and doctor would make an important change in both patient and physician education. Involving the patient in the decision-making process, if at first only as an observer, would have the important effect of increasing his understanding of why particular diagnostic and treatment procedures are being initiated or stopped. This would improve compliance and might diminish lawsuits against physicians, a symptom of failed communication. But it would also be educational for the physician, in that he would be constantly reminded of personal factors about his patients to be considered.

The effect of therapeutics might also be improved. In the *CMAJ* P. Biron writes "In man the total efficacy of drugs results partly from a pharmacologic effect, related directly to the plasma concentration, and partly from a placebo effect, related directly to the patient-doctor relationship. These two determinants are added rather than multiplied." If we accept that access to medical records, and subsequent discussion, would strengthen the doctor-patient relationship, then drugs and other procedures might increase in therapeutic effectiveness.

With potentially fatal illnesses, many physicians feel patients may be better off not knowing about certain aspects of their situation. However, to deny information, particularly to the interested patient, deprives him of the final gram of human dignity in facing death.

In the words of Elizabeth Kubler-Ross: "They all sensed a change in attitude and behavior when the

the nature of their task, physicians are far more obsessed about eliminating our possibilities, than patients could ever profitably become. Reading and discussing a differential diagnosis could be a rather overwhelming task. A sense of perspective takes many years of medical training to develop and is very difficult to communicate. The time spent explaining esoteric lab data and allaying fears might more wisely be spent on other aspects of the relationship.

In addition, people may never forget the possibility of an illness they saw referred to in their chart, though they forget the impact of your discussion. Apparently quite well satisfied with your explanation of why carcinoma was so far down on the differential diagnosis, a patient may always remember the possibility and his fear, though he has forgotten why you said he didn't have the disease.

The overwhelming of a patient with information may also be done with a single phrase: "You have a fatal illness." In the past the physician has always served as a filter, selecting what the patient learned and how he learned it, gearing the discussion to what he felt the patient could accept at the time. Some physicians have failed miserably at this. But however imperfect the system, it has the virtue of introducing information personally and gradually, with some consideration of its impact on the patient.

At the opposite end of the spectrum, the patient with lifestyle diseases is a persuasive candidate for reading his own chart. Reading and accepting this report on his health could be an important step toward assuming new responsibility for his actions. Yet what does he gain from reading his chart, that he would not gain simply from being told his blood pressure and other findings? More important is the time spent on non-charted epidemiologic

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A multitude of alternatives are presently being contemplated. The one which appears most promising and is now being tried is loss or no-fault insurance. It would seem to provide an inexpensive and just method with dealing with malpractice cases.

Victims of medical accidents would be assured recovery without undergoing the burden of hiring lawyers and proving fault, or, in the alternative, suffering their losses. Doctors and hospitals would also be relieved of the threat and stigma of malpractice claims. Doctors would be released to engage in more progressive diagnosis and therapy.¹⁴

To deal with any repeated carelessness or obvious negligence by doctors, the disciplinary committees within the medical profession's governing body would have to be given greater authority.

Continuing education and reassessment have also been suggested as a means by which the risk of malpractice could be reduced.

Admittedly, the above analysis has been rather superficial treatment of a very complex subject. However, it is hoped that the preceding review **will** serve to stimulate further thought and discussion of this most troublesome issue.

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diagnosis of malignancy was made and became aware of the seriousness of their condition because of the changed behavior of the people in their environment. In other words, those who were not told explicitly knew it anyway from the implicit messages or altered behavior of relatives or staff."

While not definitive therapy for the professions' difficulties, allowing the patient access to his treatment record is a positive step towards resolving several intricate problems.

Jeff Edelson

Con (continued)

information, the evidence that the disease was of his own choosing and may be forestalled by a change in his behavior. Once again, data are less important than perspective.

Several very different questions would have to be answered before a decision could be made to give patients access to their records. For instance, what would be the effect of changing the physician's traditional status to the less omnipotent role of technical advisor and counsellor? Desirable in some senses, might this mean that some important aspects of practice--like the placebo effect, and the effectiveness of counselling--would be lost too? In a different vein, who would decide which patients are too psychotomic, depressed or irresponsible to be offered access to their records?

Time, money, paperwork, security considerations and a vast increase in lawsuits are only a few of the practical problems that would have to be dealt with. If the net effect of the change were likely to be beneficial, all of these could be tolerated. But it seems that the primary problem, of which the request to see records is only a symptom, is failure of the doctor-patient relationship. Treating the symptom is not likely to cure the disease, and the side effects are prohibitive.

Molly Wills

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Alcohol and Drug-Related Deaths in Toronto and Vancouver: A Comparison

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The use of data from coroners' records as an additional source of information on drug use and drug-related problems in Canada is discussed. Following a consideration of certain methodological problems, descriptive data from coroners' files in Metropolitan Toronto and Vancouver are presented. Alcohol, alone and in combination with other drugs, was involved in approximately 70% of the reported cases. A relatively high proportion of older males were among the cases examined. Further data and analysis examines the "addiction status" of the deceased, the classification of the deaths by coroners and the relationship of drug use to death.

In its 1973 *Final Report*, the Commission of Inquiry into the Non-Medical Use of Drugs (the Le Dain Commission)¹ notes a need for ongoing collection of indirect measures of drug use as a check on other direct sources of data on the use and abuse of drugs in Canada. This reflects a concern with the extent and distribution of non-medical drug use and drug-related problems. Moreover, reliable and efficient measurement, assessment and monitoring of changes in the extent, distribution and patterns of use and problems is considered crucial.²

Epidemiological data on drug and alcohol-related deaths, based on the contents of coroners' files, is an interesting yet problematic* source of

information on the extent of drug use, the characteristics of drug users, and the nature of deaths associated with drug-taking behaviors.³ Such data are used in the study of addict^{4,5} drug user⁶ and alcoholic^{7,8} populations. Drug death data also provide a basis for the study of the prevalence of alcohol and drug use and the extent of availability of alcohol and drugs in the general population.^{9,10,11,12} Attention is focused, in this regard, on licit as well as illicit substances.^{13,14}

Awareness of certain methodological problems associated with the validity and reliability of coroners' file data is essential (see 11). The principal functions of a coroner are to report on the "identity" (the personal characteristics) of persons who die under specified circumstances and to ascertain why death occurred at a specific point in time (the causal factors). As regards alcohol and drug-related deaths in particular, there are limitations on a coroner's ability to perform accurately these duties. The isolation of a drug as a relevant factor in death is often, but not always, based on a post-mortem examination. In the absence of such an examination, the validity of an alcohol or drug-related designation is suspect. However, even with a post-mortem examination there are sources of error and inherent limitations in isolating a cause of death.¹⁵ It is also important to look beyond basic toxicological findings.¹⁶ Coroners necessarily exercise discretion in assigning the label "drug addict" or "alcoholic" to a deceased person and in classifying a death as natural, accidental, suicidal or homicidal.^{17,18} The sensitivity of

a coroner or a pathologist to drugs, combinations of drugs and factors associated with drug use influences the accuracy of the reporting process.^{19,20,21} Coroners and pathologists rely on the results of police investigations that vary in informational content and comprehensiveness. Additional variation is introduced by the inferences that police officers draw from physical clues, events and statements of family members, acquaintances and witnesses.

Along with limits to a coroner's ability to perform his duties, there is a variation in the extent to which coroners in fact perform duties that are within their capabilities. While some coroners provide detailed information on the deceased and the cause of death, others tend towards brevity in reporting. Given these considerations, coroners' records best serve as a secondary source of data on: 1) the distribution of alcohol and drug use and problems in Canada in terms of personal characteristics of users; and 2) the descriptive factors associated with alcohol and drug-related deaths.

Method

Data was collected from the records of 18 full-time and part-time coroners who operated in and around the Metropolitan Toronto area in 1973. The original selection process involved matching a numbered index card, coded under the name of the coroner who supervised the investigation or inquest, with the corresponding coroner's file. Cases

* Several of these problems are discussed in this paper. For a more thorough consideration see: Leon, J.S., *Methodology and Law: Problems Associated with Coroners' Data on Alcohol and Drug-Related Deaths* (1977), forthcoming.

from outside Metropolitan Toronto and cases that did not mention alcohol or drugs were eliminated. Data from the remaining 324 "drug-related" files were recorded on a pre-code data sheet. Five of the 18 coroners files account for 58.1% of the total number of drug-related cases. Geographic area of operation and individual sensitivity to drug-related factors may be of explanatory relevance in this regard.

Following the system proposed by Letteiri and Backenheimer¹⁹, the cases are classified as being either directly or indirectly related to alcohol or drugs. "Directly-related" deaths involve drug use as a necessary and sufficient condition to account for death; that is, death would not occur if the drug were not present. "Indirectly-related" deaths involve drug use as a necessary but not sufficient condition; that is, drug use is a factor concomitant to death, but not sufficient in itself to "cause" death.

The 1973 Toronto data is compared to data collected using a similar methodology in Vancouver for the period 1970-1973.^{22*} Descriptive data from the Vancouver study for 1973 are presented separately. Limitations on the comparability of these data are attributable to: differences in the organization and the procedures of the two coroner systems; differences in the sources and the nature of information included in the coroners' files; and the differences in the classification decisions made by the coroners and the researchers in the two cities. Effects of these differences are discussed below.

Results and Discussion

Table I addresses the distribution of alcohol and drug-related deaths in terms of the personal characteristics of coroners' cases. Drug-related deaths tend to occur most frequently among those in the older age categories: 62.0% of the Toronto

* This finding of fewer narcotics addicts in Toronto is consistent with other sources of Canadian data (see †, pp. 678-683).

TABLE I PERSONAL CHARACTERISTICS ASSOCIATED WITH ALCOHOL AND DRUG-RELATED DEATHS

		TORONTO (1973)	VANCOUVER (1973) (1970-1973)	
AGE	under 30	18.2	19.	19.
	30-44	19.8	27.	27.
	45 and over	62.0	54.	54.
	TOTAL (N)	100.0 (324)	100. (250)	100. (987)
SEX	male	71.	--**	71.
	female	29	--	29.
	TOTAL (N)	100.0 (324)		100. (987)
MARITAL STATUS	single	24.4	36.	39.
	married***	27.2	41.	39.
	divorced	14.8	15.	12.
	separated	8.0	7.	9.
	no information	25.6	1.	1.
	TOTAL (N)	100.0 (324)	100. (250)	100. (987)
EMPLOYMENT STATUS	currently employed	23.5	--**	63.
	currently unemployed	22.2	--	4.
	housewife	8.0	--	18.
	retired	11.7	--	4.
	student	2.2	--	4.
	no information	32.4	--	8.
	TOTAL (N)	100.0 (324)	--	100.* (987)
ADDICTION STATUS	narcotic-addict	1.5	18.	17.
	alcoholic	41.4	22.	27.
	non-addict	52.5	53.	45.
	undetermined	1.6	7.	11.
	TOTAL (N)	100.0 (324)	100. (250)	100. (987)

* Percentage total is rounded to 100.0.

** No data available.

*** Includes "common" relationships.

cases and 54% of the Vancouver cases were 45 years of age or over. A higher proportion of the Vancouver (27%) than the Toronto cases (19.8%) are in the intermediate 30-44 years category. In both cities drug-related deaths also tend to occur more frequently among males (71%) than females (29%).

The Toronto data on marital status is based primarily on the police "Sudden Death and Homicide Report" contained in the coroners' files. In over one quarter of the files, no information is available. In other files the police reports are, on occasion, somewhat ambiguous. There is no indication that these problems are encountered in the Vancouver data. The highest proportion of 1973 cases in Toronto (27.2%) and Van-

couver (41%) are recorded as married, followed closely by those who are single (24.4% in Toronto; 36% in Vancouver). Over the four year study period in Vancouver an equal proportion (39%) were married and single. Thus, drug-related coroners' cases could not be meaningfully distinguished in terms of marital status.

The Toronto data on employment status is likewise deficient both in quality and quantity. There is no information available of the Toronto cases and 8% of the Vancouver cases. While in both cities a higher proportion of the drug-related deaths are classified as currently employed (23.5% in Toronto; 63% in Vancouver) than currently unemployed (22.2% in Toronto; 4% in Vancouver), the difference between

the two categories is far greater in Vancouver than in Toronto. The Vancouver study notes a tendency on the part of coroners to report ambiguous cases as employed. An opposite tendency is suggested for Toronto. Given the problems associated with data on marital status and employment status, these variables are not used in further analysis.

Coroners frequently exercise discretion in labelling a deceased as a narcotic addict or an alcoholic. This decision is apparently based on observation, or on information provided by police officers, toxicologists, pathologists or friends and relatives of the deceased. The validity and reliability of these labels, particularly the alcoholic label, is questionable. The relevant sources of information in a given case are often not stated and the accuracy of this information is rarely assessed. In the absence of a conclusive finding of alcoholism or addiction by the coroner, further discretion is exercised by researchers in coding certain cases as undetermined. Subject to these limitations, in 1973, 1.5% of the Toronto cases and 18% of the Vancouver cases are labelled narcotic addicts.* In comparison, 41.4% of the Toronto cases and 22% of the Vancouver cases are labelled alcoholics. The highest proportion of cases in Toronto(52.5%) and Vancouver(53%) evidenced no or insufficient indication of addiction or alcoholism.

Data on selected factors characteristic of alcohol and drug-related deaths appear in Table 2. In contrast to Toronto, where 31.8% of the coroners' cases are classified as directly-related to drugs and alcohol and 68.2% are classified as indirectly-related, 73% of the 1973 Vancouver cases are classified as directly related and 27% as indirectly related. This difference at least in part, may be attributed to the discretion exercised by coroners and others in the investigative process. In isolating the direct cause of death, the assignment of casual significance to alcohol, as opposed to another disease, is often arbitrary. In 34.2%

* There are no significant differences in the number of drug-related deaths in any of the four years.

of the 1973 Toronto drug-related coroners' cases, the direct cause of death is one of the following diseases associated with excessive alcohol consumption: cirrhosis of the liver, heart disease, broncholar pneumonia or intra-abdominal hemorrhage. Only 8% of the Vancouver cases specify these diseases as the direct cause of death. Also, because the coroners' recording systems do not sopecifically include a direct-indirect classification of drug-related deaths, further variability is introduced by the ex-post facto categorization of cases by researchers.

A death is classified by an investigating coroner as being natural(no "complicating" factors), accidental("unintentional" circumstances), suicidal or homicidal.

However, the circumstances of a death are not always clear and coroners may be influenced in their decisions by extraneous factors.^{17,18} A further category, "ambiguous", is added as a check on this decision making process. Of the Toronto cases: 43.2% are classified as natural deaths; 24.7% as accidental deaths; 21.3% as suicidal deaths; 4.3% as homicidal deaths; and 6.5% as ambiguous. The 1973 Vancouver data bear no similarity to the Toronto data with regard to natural(2%) and accidental(57%) deaths. In part, this divergence reflects the discretionary element involved in classification. The Vancouver figures for suicidal(30%), homicidal(3%) and ambiguous(9%) deaths are more comparable to the Toronto figures.

TABLE II FACTORS ASSOCIATED WITH ALCOHOL AND DRUG-RELATED DEATHS

	TORONTO (1973) %	VANCOUVER (1973) % (1970-1973)	
RELATIONSHIP OF DRUG USE TO DEATH			
direct	31.8	73.	67.
indirect	68.2	27.	33.
TOTAL (N)	100.0 (324)	100. (250)	100. (987)
CLASSIFICATION OF DEATH			
natural	43.2	2.	9.
accidental	24.7	57.	52.
suicidal	21.3	30.	26.
homicidal	4.3	3.	3.
ambiguous	6.5	9.	11.
TOTAL (N)	100.0 (324)	100.* (250)	100.* (987)
GENERAL CATEGORIES OF DRUGS INVOLVED			
narcotics	1.5	5.	4.
barbiturates	7.1	11.	12.
non-barbiturate sedative-hypnotics	1.5	2.	1.
alcohol	61.7	43.	45.
tranquilizers/ antidepressants	3.0	0.	1.
analgesics	2.5	1.	1.
alcohol & barbiturates	6.8	15.	13.
alcohol & analgesics	1.5	2.	1.
alcohol, narcotics and barbiturates	0.0	2.	3.
alcohol, narcotics	0.0	6.	8.
narcotics, barbiturates	0.0	4.	4.
drugs - not specified	1.5	0.	0.
other	12.6	9.	7.
TOTAL	100.0 (324)	100. (250)	100. (987)

* Percentage total is rounded to 100.%,

The general category of drug most frequently implicated in the 1973 coroners' cases is alcohol(61.7% in Toronto; 43% in Vancouver). Barbiturates alone(7.1% in Toronto; 11% in Vancouver) and in combination with alcohol(6.8% in Toronto; 15% in Vancouver) are, relative to the other drug categories, also frequently involved. Narcotics alone(5%) and in combination with alcohol(6%) and barbiturates(4%) are, as might be anticipated from the figures in narcotic addiction in **Table I**, more evident in Vancouver's drug-related deaths than in Toronto's.

Further analysis of the addiction status assigned to alcohol and drug-related coroners' cases(**Table III**) indicates that those labelled narcotic addicts tend to be in the younger age categories, with their deaths being classified as accidental and directly related to drug use. The small number of Toronto narcotic addicts renders it difficult to meaningfully interpret the Toronto data and may account for the higher proportion of females than males in this group. The Vancouver data indicating that 76% of the addicts are male is more credible. Alcoholics tend to be male and in the 45 years and over category.* Holding sex constant, 81.8 % of the male alcoholic cases and 82.3% of the female alcoholic cases in Toronto and 76% of the male alcoholic cases and 70% of the female alcoholic cases in Vancouver are 45 years of age or over. In Toronto, the alcoholic cases tend to be classified as natural deaths(81.3%) and indirectly drug-related(88.1%). In Vancouver they tend to be classified as accidental(52%) or to a lesser extent natural(24%), and a slightly higher proportion are deemed directly(54%) rather than indirectly(46%) drug-related. The discretionary factors associated with these two variables are noted above. Those cases in the non-addict category also tend to be older and male, although to a lesser extent than the

* This pattern held for all Toronto alcohol-related deaths: 72.5% of the 200 cases are 45 years of age or over and 82% are male. Also, 91.5% of these cases are classified as being indirectly-related to drugs.

TABLE III ANALYSIS OF ADDICTION STATUS LABELS APPLIED TO ALCOHOL AND DRUG-RELATED CORONERS' CASES

	NARCOTIC-ADDICT		ALCOHOLIC		NON-ADDICT		UNDETERMINED	
	TORONTO (1973)	VANCOUVER (1970-1973)	TORONTO (1973)	VANCOUVER (1970-1973)	TORONTO (1973)	VANCOUVER (1970-1973)	TORONTO (1973)	VANCOUVER (1970-1973)
AGE								
under 30	60.0	49.	1.5	4.	31.2	18.	6.7	10.
30-44	40.0	37.	16.4	21.	19.4	27.	46.7	31.
45 and over	0.0	14.	82.1	75.	49.4	55.	46.7	59.
TOTAL (N)	100.0 (5)	100. (169)	100.0 (134)	100. (263)	100.0 (170)	100. (446)	100.0* (15)	100. (109)
SEX								
male	40.0	76.	82.1	81.	61.8	62.	86.7	72.
female	60.0	24.	17.9	19.	38.2	38.	13.3	28.
TOTAL (N)	100.0 (5)	100. (169)	100.0 (134)	100. (263)	100.0 (170)	100. (446)	100.0 (15)	100. (109)
CLASSIFICATION OF DEATH								
natural	0.0	0.	81.3	24.	15.3	4.	33.3	6.
accidental	100.0	89.	9.0	52.	32.4	36.	53.3	58.
suicidal	0.0	4.	7.5	13.	33.5	45.	13.3	15.
homicidal	0.0	2.	0.0	0.	8.2	3.	0.0	6.
ambiguous	0.0	4.	2.2	11.	10.6	13.	0.0	16.
TOTAL (N)	100.0 (5)	100.* (169)	100.0 (134)	100. (263)	100.0 (170)	100.* (446)	100.0* (15)	100. (109)
RELATIONSHIP OF DRUG USE TO DEATH								
direct	80.0	95.	11.9	51.	47.6	66.	13.3	61.
indirect	20.0	5.	88.1	46.	52.4	34.	86.7	39.
TOTAL (N)	100.0 (5)	100. (169)	100.0 (134)	100. (263)	100.0 (170)	100. (446)	100.0 (15)	100. (109)
GENERAL CATEGORY OF DRUGS INVOLVED								
narcotics	40.0	20.	0.0	0.	0.0	0.	0.0	1.
alcohol	0.0	4.	90.3	75.	41.8	40.	53.3	57.
barbiturates	0.0	3.	0.7	3.	12.4	23.	6.7	6.
alcohol and barbiturates	0.0	4.	2.2	11.	10.0	17.	13.3	12.
other	60.0	69.**	6.8	11.	35.8	20.	16.7	24.
TOTAL (N)	100.0 (5)	100. (169)	100.0 (134)	100. (263)	100.0 (170)	100. (446)	100.0 (15)	100. (109)

* Percentage total is rounded to 100.0%.

** Of these cases, 37% involved alcohol and narcotics, 22% involved barbiturates and narcotics and 10% involved alcohol, barbiturates and narcotics.

alcoholics.* In Toronto, 32.4% of these deaths are reported as accidental and 33.5% as suicidal. In Vancouver, 36% are reported as accidental and 45% suicides. In the non-addict category, alcohol is involved in approximately 40% of the cases in both cities.

Analysis of the relationship of drug use to death(**Table IV**) highlights the predominance of older cases(67.9% in Toronto; 60% in Vancouver), males(79.6% in Toronto; 85% in Vancouver) and alcohol; (82.8% in

* In comparison to the Toronto figures for alcohol-related deaths, 43.5% of the 23 drug-related deaths involving barbiturates alone and 54.5% of the deaths involving barbiturates in combination with alcohol are 45 years of age or over. 52.2% of the barbiturate cases and 59.1% of the combination cases are male. Also, 73.9% of the barbiturate cases and 63.6% of the combination cases are classified as directly-related to drugs.

Toronto; 89% in Vancouver) in the drug-deaths categorized as indirectly-related. With regard to the classification of drug-related deaths(**Table V**), the relatively less prominent role played by alcohol in suicidal deaths(20.3% in Toronto; 28% in Vancouver) is apparent. As might be expected, a high proportion of the cases classified as natural deaths in both Toronto(78.6%) and Vancouver(81%) are in the "45 and over" age category. The suicidal, homicidal and ambiguous cases involve an approximately equal proportion of males and females.

Summary and Conclusions

Descriptive data collected from Toronto and Vancouver coroners' files indicates a relatively high proportion of males among those whose deaths are directly or indirectly related to alcohol and drug use. Alcohol and

alcoholism figure predominantly in these cases. The significance of alcohol is even more apparent when the probable under-reporting of alcohol-related deaths is considered. Inter-city differences in the classification of death and in the relationship of drug use to death can be attributed to variations in discretionary decision-making and interpretation. The limitations on the validity and reliability of coroners' file data should not be underestimated. Still, this data does add a further dimension to the study of drug use and drug-related problems in Canada.

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TABLE IV RELATIONSHIP OF DRUG USE TO DEATH IN CORONERS' CASES

	DIRECT %		INDIRECT %	
	TORONTO (1973)	VANCOUVER (1970-1973)	TORONTO (1973)	VANCOUVER (1970-1973)
AGE				
under 30	27.2	19.	14.0	18.
30-44	23.3	30.	18.1	22.
45 and over	49.5	51.	67.9	60.
TOTAL (N)	100.0 (N=103)	100. (N=664)	100.0 (N=221)	100. (N=323)
SEX				
male	52.4	64.	79.6	85.
female	47.6	37.	20.4	14.
TOTAL (N)	100.0 (N=103)	100.* (N=664)	100.0 (N=221)	100.* (N=323)
GENERAL CATEGORIES OF DRUGS INVOLVED				
alcohol	16.4	24.	82.8	89.
barbiturates	16.4	16.	2.7	6.
alcohol & barbiturates	13.6	19.	3.6	1.
other	53.6	41.	10.9	4.
TOTAL (N)	100.0 (N=103)	100. (N=664)	100.0 (N=221)	100. (N=323)

* Percentage total is rounded to 100%.

TABLE V CLASSIFICATION OF ALCOHOL AND DRUG-RELATED CORONERS' CASES

	NATURAL %		ACCIDENTAL &		SUICIDAL %		HOMICIDAL %		AMBIGUOUS %	
	TORONTO (1973)	VANCOUVER (1970-1973)	TORONTO (1973)	VANCOUVER (1970-1973)	TORONTO (1970)	VANCOUVER (1970-1973)	TORONTO (1973)	VANCOUVER (1970-1973)	TORONTO (1973)	VANCOUVER (1970-1973)
AGE										
under 30	4.3	7.	30.0	23.	26.1	19.	28.6	36.	33.3	9.
30-44	17.1	17.	18.8	28.	26.1	27.	28.6	44.	14.3	28.
45 and over	78.6	81.	51.3	49.	47.8	54.	42.9	20.	52.4	63.
TOTAL (N)	100.0 (140)	100. (88)	100.0* (80)	100.0 (510)	100.0 (69)	100. (254)	100.0* (14)	100. (25)	100.0 (21)	100. (110)
SEX **										
male	80.7	-	75.0	-	55.1	-	50.0	-	57.1	-
female	19.3	-	25.0	-	44.9	-	50.0	-	42.9	-
TOTAL (N)	100.0 (140)	-	100.0 (80)	-	100.0 (69)	-	100.0 (14)	-	100.0 (21)	-
GENERAL CATEGORIES OF DRUGS INVOLVED										
alcohol	88.5	93.	58.8	52.	20.3	28.	71.4	68.	23.8	13.
barbiturates	1.4	5.	6.3	7.	15.9	26.	0.0	0.	23.8	15.
alcohol and barbiturates	2.1	0.	10.0	8.	8.7	14.	7.1	4.	19.1	44.
other	8.0	2.	24.9	33.	55.1	32.	21.5	28.	33.3	28.
TOTAL (N)	100.0 (140)	100. (88)	100.0 (80)	100. (510)	100.0 (69)	100. (254)	100.0 (14)	100. (25)	100.0 (21)	100. (109)

* Percentage total is rounded to 100%.

** No data were available for classification of death by sex for Vancouver.

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Any staff member wishing to purchase a copy of the first Medical Yearbook can send in a request with an accompanying cheque for \$7.50 made out to: **The Medical Society, c/o the Yearbook Committee.** Orders must be placed before December 15, 1977, as only a limited number of copies are available.

R.I. Harris Prize

The Editors are pleased to announce that J.D. Gollish, 7T8, has won the R.I. Harris prize for his article *Prognostic Indicators in the Treatment of Ankle Fractures*, May, 1977. This prize is awarded annually by the Orthopaedic Surgery Committee to the author of the article judged the best written on a topic related to orthopaedic Surgery. It has a monetary stipend of \$100.

Webster Prize

The winner of the 76/77 Webster Prize is Peter Dobson, 7T9, for his article, *Crohn's Disease*, December, 1976. This award, granted annually by the Academy of Medicine, carries a \$75 stipend plus one year's membership in the Academy. It is awarded for the best overall article to appear in the Journal each year, and is decided on the basis of both style and content.

The Journal wishes to congratulate the winners and thank the Orthopaedic Surgery Committee and the Academy of Medicine for their continued interest in undergraduate journalistic and scholastic efforts.

FEEDBACK

Dear Editor;

I am very impressed with the new U. of T. Medical Journal and I think you've done an excellent job of rejuvenating it. I send this (financial support) in hopes that you and your staff will be encouraged to maintain your high standards.

Sincerely,
Dr. J. Weir
Halifax
July, 1977

(continued from page 12)

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Book Reviews

The Psychological Problems of the Child and His Family

Paul D. Steinhauer and Quentin Rae-Grant

Macmillan of Canada

PUBLISHED: April 1977

PRICE: \$12.95

This book is a new release from the Hospital For Sick Children. Its purpose is to present a comprehensive, informative discussion of child psychiatry and at the same time emphasize the basics and keep the text understandable to a reader new to the discipline.

The authors have succeeded in producing a manual which is clear, organized, sequential, easy to read and enjoyable. Each chapter has an excellent step by step explanation of new ideas. There are helpful diagrams and clinical vignettes which illustrate the topic being discussed. The child is seen as an individual with his own drives and defenses and a member of a family and society. There is emphasis on the process of personality development in a child and the consequences of blocks and deviations to this development.

The book is organized in a logical, easy to follow format. The first two units deal with what is expected in normal personality development

through childhood and adolescence, and the normal interaction between child and family. There is then a practical guide for the primary care physician to the assessment of a child or family problems.

Unit Three examines the syndromes seen in child psychiatry. Only the common ones are discussed so no pages are used on rare presentations. The best chapter in the book is "Psychoneurosis, Behavior Disorders and Personality Disorders in Children". It is presented systematically from first principles on a level that anyone could understand. There are excellent diagrams and illustrative case histories. Management of these problems by non-psychiatrists is clearly presented. There are very practical chapters on such common problems as managing the family of a retarded child and preparing a child to visit the dentist.

Unit Four deals with crisis situations in a child's or family's life, such as hospitalization, death, battering or divorce.

Unit Five gives an overview of the treatment of children and families with psychological problems. The survey of drug therapy, indications, dosages and side effects is very useful.

The greatest strength of this text is

its practical approach. Each chapter can be read independently and gives the reader a complete picture of how the illness has developed, how it can be recognized and the best way to treat or prevent it. Indications for psychiatric referral are also discussed.

The book is clearly directed to the primary care physician who wants a fast but complete reference, and the student who needs a basic systematic introduction to child psychiatry.

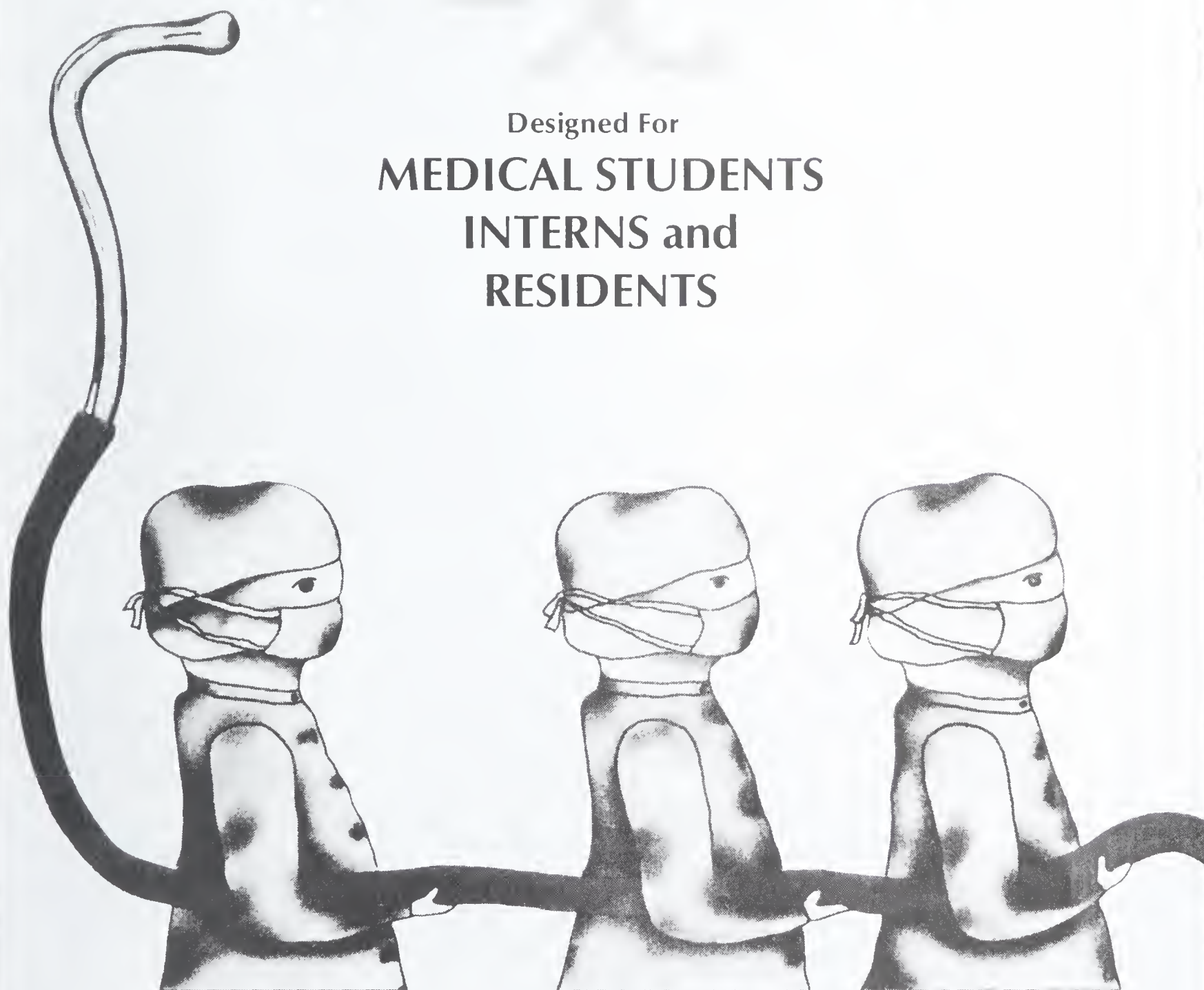
Grant W. Mullen, 7T9

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